

SCHISTOSOMIASIS JAPONICA

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1. GENERAL. Three species of blood flukes are important agents in the production of human disease *Schistosoma haematobium*, *S. mansoni*, and *S. japonicum*. These parasites belong to the class of trematodes in the Phylum Platyhelminthes or flatworms. The three species have similar life cycles, but their respective molluscan intermediate hosts are different. Man becomes infected by each species in much the same way. After reaching maturity, *S. haematobium* characteristically settles in the venous plexuses of the urinary bladder. In maturity, both *S. mansoni* and *S. japonicum* inhabit branches of the superior and inferior mesenteric veins. The diseases produced by these flukes have essentially the same pathogenesis, and differ chiefly because of variations in the anatomical regions involved and consequent variations in symptomatology. The same symptoms may appear in all three infections during the early stages. The disturbances produced by *S. haematobium* arise mainly in the genito-urinary system, but the lower colon and rectum also occasionally may be involved. Both

S. mansoni and *S. japonicum* cause disturbances principally in the small intestine, colon, and rectum; rectal lesions are less commonly due to *S. japonicum*. All three parasites may give rise to hepatic lesions, but severe hepatic cirrhosis, splenomegaly, and ascites are common late results of infection with *S. mansoni* and more especially with *S. japonicum*. In general, infection with *S. japonicum* is more severe and appears to be more resistant to available chemotherapy than is the case with disease due to the other blood flukes. The available methods of treatment can be applied without variation to any of the three forms of schistosomiasis. In a broad sense, the same methods of prevention are applicable. Apart from these generalities, the remainder of this bulletin is devoted to infections with *S. japonicum*, or schistosomiasis japonica. This infection is sometimes referred to as oriental schistosomiasis or as Katayama disease (from the name of one of the Japanese areas in which it occurs).

2. GEOGRAPHIC DISTRIBUTION. *a. General.* The extent of schistosomiasis japonica is determined principally by the distribution of

the species of snails which serve as intermediate hosts. The established occurrence of the disease is limited to Japan, Formosa, China, the

Philippines, and Celebes; foci may exist in upper Burma along the Chinese border. (See fig. 1.)

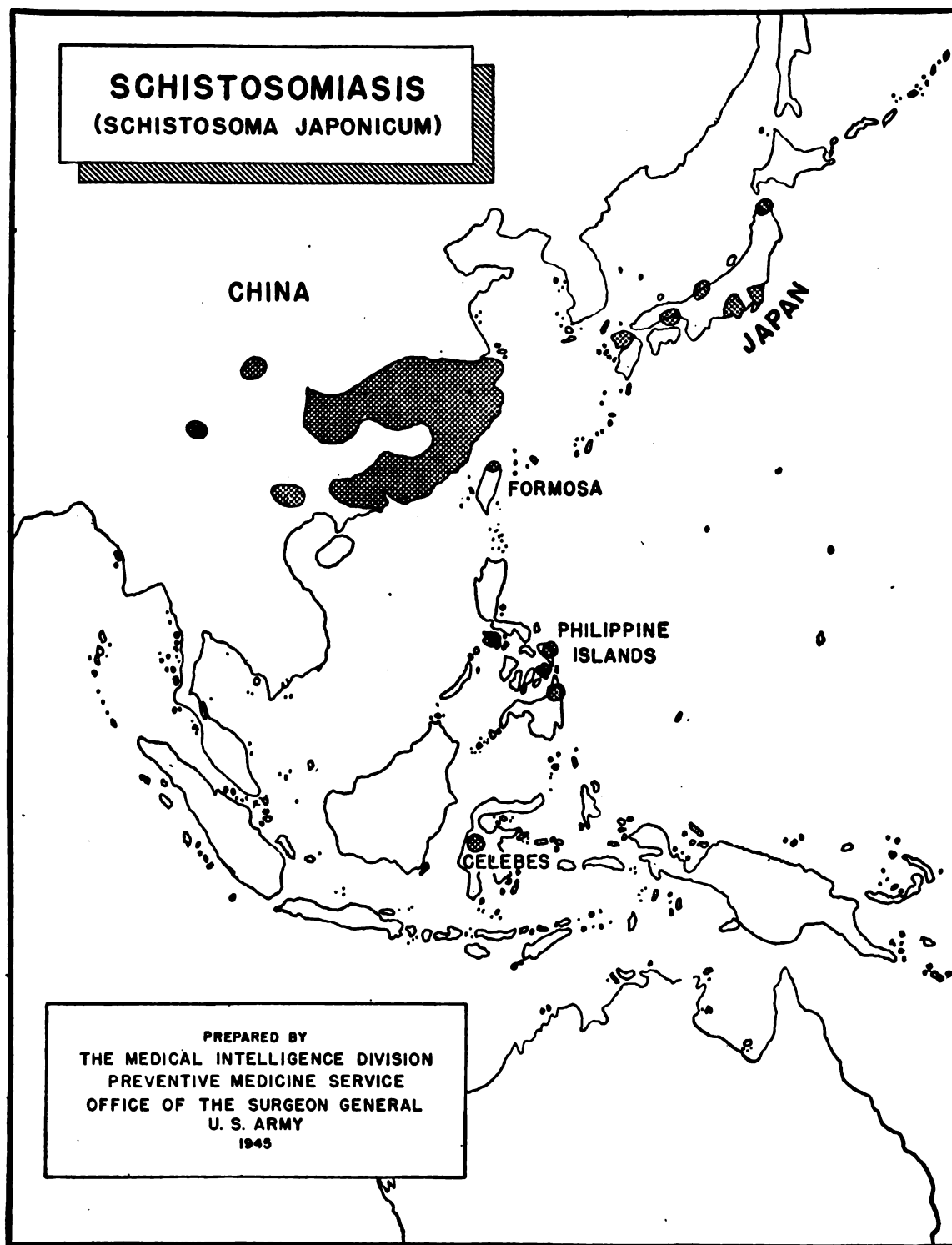


Figure 1.

b. Japan. Five foci are well known. These involve small areas, but at least in the past the rate of infection has been high. Four of these foci are on the main island of Honshu: In the prefecture of Ibaraki, on the lower reaches of the Tone River, not far to the northeast of Tokyo; in the prefecture of Shizuoka, about Lake Ukishima, near Numadzu; in the prefecture of Yamanashi, near Kofu, north of Mt. Fuji; in the prefectures of Okayama and Hiroshima, along the Ashida River in the Katayama district. The fifth focus is on Kyushu, the southern island, in the prefectures of Saga and Fukuoka, along the Chikugo River, near Kurume. In addition, reports of surveys show individuals passing schistosome eggs in the prefectures of Fukui, Tochigi, and Aomori, on the Island of Honshu. According to a recent unconfirmed report, schistosomiasis exists on the Island of Shinkoku.

c. Formosa. According to one report, a focus exists near Shinchiku, in the northwest part of the island. It is possible that schistosomiasis occurs elsewhere on the island.

d. China. Schistosomiasis is widely distributed in central and southern China, where very large numbers of people are infected. It has not been found north of the tributaries of the Yangtse River. In all probability, the distribution of schistosomiasis in China is wider than is yet realized.

(1) *Southern areas.* Schistosomiasis is found in Kwangsi Province in the valley of the Si River (region of Pinyang). Kwangtung has endemic areas on the Si River (about Takhing or Tehking), on the Pei River (area of Kukiang or Shiuchow), and on the Han River (Meihsien). Fukien has areas about Futsing and Minhow (Foochow). Chekiang includes areas about Yungkia (Wenchow) and Taichow and extensive districts about Hangchow Bay (including Ningpo, Shaohsing, and Kiahsing) and in the Chientang River basin from Hangchow to Chuhsien and Kaihwa. Chekiang and Kiangsu together present a heavily infected area about the Tai Lake.

(2) *Yangtse River areas.* Schistosomiasis occurs in areas which are thickly scattered throughout the drainage basin of the Yangtse, especially on its southern side, from the outskirts of Shanghai (Kiangsu Province), near its mouth, to Ichang in Hupeh Province. The

infection is especially prevalent in the Hankow-Wuchang area (Hupeh). It is also prevalent about several large lakes and their tributaries and effluents, including Chao Lake (Anhwei Province), Poyang Lake (Kiangsi), and Tungting Lake (Hunan). West of Ichang, the disease has been reported only along the Min River south of Chengtu.

(3) *Yunnan.* An endemic area exists in the region of Tali on the Lake Erh-hai. Cases have also been reported on the Burmese border.

e. The Philippines. Schistosomiasis has been reported to occur especially on the Island of Leyte, and also on Samar, Mindoro, and in the region of Surigao in the northern part of Mindanao.

f. Celebes. A focus has been reported in the Lake Lindoe region in the central portion of the island.

3. ETIOLOGIC AGENT. *a.* Unlike other trematodes, the adult worms of *Schistosoma* have separate sexes. The female worms of *S. japonicum* are long and slender, measuring about 2.5 cm by 0.03 cm. The males are shorter and thicker; the sides of the posterior part of the body curl ventrally to form a groove in which the female worm is enclosed during most of her life. The female deposits eggs in the small venules of the intestinal wall. Some of these eggs gradually are extruded into the lumen and are discharged in the feces.

b. Since the eggs are usually fully developed when passed, they hatch within a few hours upon dilution of the feces with fresh water, liberating a ciliated free-swimming larva known as a miracidium. Upon reaching a suitable snail which can serve as intermediate host, the miracidium invades the soft tissues of the mollusc where it undergoes development and asexual multiplication requiring several weeks. The ultimate result is the production of numerous free-swimming, fork-tailed cercariae, the infective stage for man.

c. Cercariae emerge from infected snails over a period of several months. The cercariae are microscopic in size, their bodies measuring only about 0.15 mm in length. They tend to collect near the surface of the water. They are short-lived and must reach their final host within 1 or 2 days in order to continue their development. If cercariae are successful in contacting the skin or mucous membrane of man, or appropriate

animals, they drop their tails, rapidly penetrate the tissue, and enter the venous circulation. After traversing the lungs and heart, the young worms that reach the intra-hepatic portions of the portal vein mature and pair. They then proceed against the blood stream to small branches of the mesenteric veins where they settle down. Those that settle in small vessels elsewhere for the most part are unable to complete their development, die, and are absorbed. Mature worms are sexually paired in the mesenteric venules where egg deposition occurs. Eggs first appear in the feces from 4 to 6 weeks following invasion of the body by cercariae. In the absence of effective treatment, adult worms may live and continue to deposit eggs for many years (perhaps 10 or 20 years).

4. TRANSMISSION. *a.* Schistosomiasis is most prevalent in irrigated regions where the abundance of canals, ditches, and pools of stagnant water provide a favorable environment for the snail intermediate hosts. Only certain species of snails belonging to the genera *Katayama*, *Oncomelania*, and *Schistosomophora* are suitable for the larval development of *Schistosoma japonicum*. Sources of infection are limited to regions where these species are present. The snails are amphibious in their habits and because of their small size (3 to 10 mm in length) may easily be overlooked unless careful search is made of vegetation above the water line. They apparently prefer water which is slightly acid, and are most abundant in still water and sluggish streams with grassy banks. Rapidly flowing water, however, cannot be regarded as necessarily safe, since cercariae may be washed into the stream from more favorable snail habitats located higher up. Inundated rice fields and ponds are particularly dangerous sources of infection. Infested water may be quite clear and show no gross evidence of contamination.

b. Infection is generally acquired by wading, swimming, bathing, or otherwise coming in contact with water infested with cercariae. Contaminated drinking water is another but apparently less important source of infection. Cercariae consumed in drinking water presumably enter the tissues by penetrating the buccal mucosa. A few minutes of contact with heavily infested water are sufficient to produce a clinical

infection. Ordinary clothing does not significantly impede penetration of the cercariae into the skin.

c. Spread of schistosomiasis is favored by poor sanitation which allows contamination of water with feces containing eggs. Use of human excrement to fertilize rice fields is an important factor in the prevalence of schistosomiasis. However, animals other than man may also be involved in the spread of the disease. Dogs, cats, field mice, horses, cattle, and water buffalo may harbor *Schistosoma japonicum* and serve to disseminate the infection among snails.

5. COURSE. *a. General.* The lesions and symptoms of schistosomiasis japonica are due to the presence of the worms and their eggs, and probably also to substances given off by both worms and eggs. It is customary to divide the course of the disease into three stages, but these stages are continuous, and, therefore, clinical phenomena attributed to the different stages may overlap. The first stage corresponds to the period from the penetration of the body by cercariae to the settling down of paired mature worms in the mesenteric venules. The second stage corresponds to the period in which eggs are deposited by female worms in the small vessels of the intestinal wall. Many of these are extruded into the lumen of the gut and appear in the feces. Others are carried in numbers, which increase as time goes on, into the liver, mesenteric lymph nodes, and to a lesser degree other abdominal organs. In unusual cases, eggs may be found in the lungs, heart, brain, or other parts of the body, including the skin. Vastly more eggs, however, are always found in the intestinal wall and liver than elsewhere. The presence of eggs in the tissues gives rise to small abscess-like formations, which, in the case of the intestine, often rupture and release their contents into the gut. In practice, the symptoms associated with the first stage may be inseparable from those of the second stage. The symptomatology of these stages, therefore, is described under one heading (*b* below). The third stage, which is associated with proliferation and repair of damaged tissues, is described in *c* below.

b. Early symptoms. The early symptoms are variable in time of appearance and in intensity.

It should be remembered that the symptoms are not necessarily a measure of the severity of the infection. Early symptoms are often transient and may be overlooked. In some cases, a papular rash or itching, or both, may appear immediately after exposure. However, the first symptoms usually are noted between 3 and 10 weeks after infection (most commonly after about 5 to 6 weeks). The earliest symptoms include fever, chills, sweats, itching, unproductive cough, headache, stiffness of neck muscles, chest pain, epigastric pain, general abdominal discomfort or crampy pain, and pain in the back or legs. (Stiffness of the neck is described as apparently of muscular origin.) Anorexia and weight loss are often striking. Diarrhea may develop somewhat later or may be present at the same time; it is not usually severe in the beginning. The picture of subacute appendicitis occasionally develops. In some instances, signs of involvement of the central nervous system appear, including confusion or stupor, muscular weakness, changes in reflexes (especially hyperactivity), and rarely epileptiform seizures. Urticaria, with large or small lesions, edema, signs suggestive of bronchopneumonia, and abdominal tenderness (especially in the epigastrium), may be present. Lymph node enlargement, either regional or generalized, may be demonstrable. The liver and, at a somewhat later stage, the spleen are often palpable. X-ray pictures of the lungs may show scattered areas of infiltration. In rare cases, peripheral thrombosis has been attributed to the local deposit of eggs. In the early stages, the blood characteristically shows a rapidly increasing white cell count, with marked eosinophilia. These changes in the blood may reach striking proportions. As time goes on, they tend to disappear. At first there is no anemia. After deposition of eggs has begun, the stools may contain blood and mucus, as well as eggs. These symptoms may last from 2 to 10 weeks. There may be relapses and remissions, but spontaneous cessation of early symptoms ultimately occurs in most cases, if reinfection does not occur.

c. Late sequelae. As time goes on, affected tissues react to the increasing numbers of eggs deposited in them by extensive proliferation and repair. The results which may develop after a number of years include: thickening of

the intestinal wall and formation of papillomata; thrombosis of mesenteric, portal, or splenic veins; cirrhosis of the liver, splenomegaly, and ascites. Emaciation may be extreme. When these lesions develop, associated symptoms usually appear only 3 or more years after infection. The clinical picture approximates that of Banti's syndrome. Dysentery may appear from time to time, but is often absent. At this stage, frequently, if not usually, the stools do not contain schistosome eggs. Anemia may be severe. The leukocytes are often reduced in number, but may be within normal limits or slightly increased. Eosinophilia is rarely present. The disease may last for many years.

6. DIAGNOSIS. *a. Clinical.* (1) In the early stage, a history of exposure is a most important clue to the diagnosis. Particularly significant is membership in a group of individuals with the same exposure and a similar clinical course. The clinical findings of outstanding significance are urticaria, itching, fever, cough, diarrhea, palpable liver, and (somewhat later) palpable spleen. The diagnosis should be confirmed by the demonstration of ova in the stools. If necessary, proctoscopy may be used as an aid in diagnosis, provided personnel trained in the use of the instrument are available. It may be possible to see beneath the normal appearing mucosa clusters of small yellowish nodules. Specimens containing ova may sometimes be obtained when ova cannot be demonstrated in the stools. Early schistosomiasis must be separated from allergic reactions to agents other than schistosomes and from many febrile diseases, such as dengue, malaria, kala azar, typhoid fever, and tuberculosis. An enlarged and tender liver may cause confusion with amebic or other forms of hepatitis, including infectious hepatitis. When the dysenteric picture is present, amebic and bacillary dysentery, and intestinal tuberculosis must be excluded. Involvement of the appendix may suggest the presence of subacute appendicitis.

(2) In the late stage, schistosomiasis japonica may be inseparable on clinical grounds from Banti's syndrome or hepatic cirrhosis from other causes. Splenomegaly is more marked in this disease than in ordinary portal cirrhosis. Late schistosomiasis japonica may resemble

kala azar or chronic malaria. These diseases, however, are often associated with febrile episodes which are rare in late schistosomiasis; they are rarely, if ever, associated directly with ascites.

b. Laboratory. (1) *General.* Specific diagnosis is established by the demonstration of the typical eggs in the stool specimen, or by allowing the eggs to hatch and identifying the miracidia. Difficulty may be encountered in finding eggs at some stages of the disease.

(a) Eggs discharged in the feces are usually mature and contain fully developed ciliated miracidia. However, many of the eggs first passed after infection may be immature. The eggs measure 70 to 100 microns in length by 55 to 65 microns in width and have well-rounded ends. They may have a light yellowish-brown color or may be hyaline. When the egg is properly oriented, a minute spinose process resembling a recurved hook extending from a depression on one side of the egg near the end is sometimes visible. In a viable mature egg, the miracidium may be seen squirming within the shell and moving its cilia in rhythmic fashion. Detail is obscured and the appearance altered in some specimens by adherent tissue remnants and fecal debris. Occasionally stools may contain eggs in which miracidia have disintegrated.

(b) The miracidium of *S. japonicum* is a very active, free-swimming organism which propels itself by means of cilia covering its surface. When observed in the water after hatching, the miracidia show a fishlike movement. A small, primitive gut is present at the cone-shaped anterior end, and on each side of this structure is a gland which apparently secretes a lytic substance. Slightly posterior to these is a second pair of glands which open laterally. Two flame cells, conspicuous because of the rhythmic movement of their cilia, are present in each side of the body. Proliferating germ balls are located in the posterior end of the larva. Use of the high power, or oil immersion lens, is required to distinguish the detailed structure of the miracidium.

(2) *Stool examination.* Several techniques may be used to detect the eggs in stool specimens. Since the eggs (or miracidia) are difficult to demonstrate in early and also very late

cases, the diagnosis of schistosomiasis should not be excluded by a single negative stool examination.

(a) *Examination of a direct smear.* A simple smear of feces mixed with a drop of physiological saline solution on a slide reveals the eggs in many specimens from heavy- and well-established infections. Smears should be made from any blood or mucus present in the stool specimen and preferably from the surface of the stool. When negative results are obtained, this technique should be supplemented by other procedures listed below. Concentration of eggs by zinc sulfate flotation is not satisfactory.

(b) *Sedimentation.* A portion of the stool specimen is comminuted in 5 to 10 parts of tap water poured into a conical urinalysis glass and allowed to sediment for 30 to 45 minutes. The supernatant fluid is decanted and the sediment resuspended by the addition of water. The process is repeated three or four times until the lighter debris has been removed and the supernatant fluid is relatively clear. After the final washing, the sediment is transferred to a slide by means of a pipette and examined for the eggs, using the low power of the microscope. Several smears should be examined before a negative report is made. Sedimentation and examination should be completed within 6 hours after dilution of the feces, otherwise hatching may occur. If eggs are not demonstrable by the sedimentation technique, an egg-hatching technique should be employed.

(c) *Egg-hatching technique.* The sedimentation technique is carried out on a generous portion of the stool specimen as described above. The final sediment is transferred to a graduate cylinder or, preferably, a large Erlenmeyer flask, and the container filled with water free from chlorine. It is allowed to stand preferably at a temperature of 30° C., for 6 to 12 hours, or overnight, to permit the eggs to hatch. The free-swimming miracidia collect near the surface of the water. The small area at the top of the flask concentrates the organisms so that they are more easily detected. The miracidia may be readily observed in proper light by use of a hand lens. They appear as minute, white, boat-shaped bodies swimming rapidly in a straight course. The miracidia must not be confused

with free-living ciliates which sometimes contaminate water. To confirm the identification, a few of the organisms should be transferred to a glass slide and examined under higher magnification. A dilute aqueous iodine stain may be added to facilitate identification.

(d) *Acid-ether technique.* Approximately 1 gram of fecal material (about the size of a pea) is thoroughly emulsified in 5 cc of 40 percent hydrochloric acid (40 cc concentrated hydrochloric acid diluted to 100 cc) in a small vial. The material is filtered through two layers of moist gauze stretched over the top of a 50 mm funnel into a 15 cc centrifuge tube. An equal quantity of ether is added, and the tube is stoppered with a gloved finger, and shaken thoroughly. It is then centrifuged for 1 minute at 1,500 rpm. Upon removal from the centrifuge, the debris floating at the acid-ether junction is loosened by ringing with a clean applicator, and the acid and ether layers are rapidly poured off and discarded. The same applicator is used to stir the sediment in the few drops of fluid remaining; the sediment is then transferred to a slide and examined under a coverslip.

(3) *Eggs in tissues.* Diagnosis sometimes may be established by the demonstration of eggs in tissue prepared by the usual techniques. The eggs are recognized by the characteristic shell. Often, however, they are collapsed, and in old infections they may be calcified.

(4) *Serological and skin tests.* Positive skin reactions appearing within 15 minutes may be obtained following intradermal injection of group-specific antigens prepared from extracts of adult schistosomes, cercariae, or infected snail tissues. Controlled skin tests, precipitin, and complement fixation reactions with schistosome antigens may be helpful as adjunct diagnostic procedures in cases in which eggs cannot be demonstrated. However, general use of these techniques in Army hospitals is precluded by the fact that the antigens are not available for distribution.

(5) *Other laboratory studies.* In the early stages, leukocytosis is often present, with total counts ranging from 12,000 to 20,000 and sometimes to 60,000 or more. Eosinophilia is common and when present is an important finding, especially when a marked increase occurs

rapidly. The presence or absence of other intestinal parasites should be determined. The percentage of eosinophils may reach 80. Except in the late stages, anemia is not necessarily present. There is usually an increase in the globulin fraction of the plasma proteins. The erythrocyte sedimentation rate is elevated in some cases. The urine is not remarkable. The stools do not show any characteristic abnormalities other than the presence of eggs, although blood and, especially, mucus are often present in the early stages. The spinal fluid has been reported as normal, even in patients with central nervous system involvement.

7. TREATMENT. a. *General.* The treatment of late schistosomiasis with established hepatic disease is not discussed here. In early schistosomiasis, if significant constitutional symptoms or diarrhea are present, the patient should be confined to bed. The diet should afford a liberal supply of all the essential nutrients. Supplementation with four multivitamin tablets a day is recommended. If diarrhea is present, the diet should be bland or soft.

b. *Chemotherapy.* (1) Drug treatment should be instituted at the earliest possible time. Compounds of trivalent antimony are the most effective known agents. Pentavalent antimony preparations, such as neostibosan, are relatively ineffectual. Most of the reported cases of schistosomiasis japonica have been treated with one of two trivalent antimony compounds, antimony and potassium tartrate or fuadin. So far as is known, no adequate comparison has been made of the efficacy of these drugs in the treatment schistosomiasis japonica. Recommended doses of fuadin, however, are less toxic than those of antimony and potassium tartrate. Other drugs have been used in the treatment of infections due to *S. haematobium* and *S. mansoni*. Available reports do not establish the superiority of any of these other drugs. Their relative value in infections due to *S. japonicum* is problematic. At present, it is recommended that patients with schistosomiasis be treated with fuadin or with antimony and potassium tartrate. The courses recommended below are planned to provide approximately equal amounts of antimony, in order to permit comparison of the two drugs on this basis. Authority for the trial use of

other chemotherapeutic agents in the United States should be obtained from The Surgeon General, or, in oversea theaters, from the theater surgeon.

(2) All available preparations of antimony possess toxic properties which are potentially dangerous. Nevertheless, the early institution of chemotherapy for schistosomiasis is imperative, unless the presence of another serious disease contraindicates it. Antimony compounds should always be given with care, especially with close observation of the patient's reactions to their administration. It should be remembered that small dosages are often followed by therapeutic failure. Among the toxic effects which have been reported are depression of the circulation and respiration, and irritation of the central nervous system, liver, and kidneys. Sudden death during injection has been reported. In general, antimony compounds are contraindicated in the presence of disease of the heart, liver, or kidneys. As a rule, they should not be administered concurrently with other metals or potential cardiac depressants, such as emetine.

c. *Fuadin*. This drug is also known as neo-antimosan and stibophan. The solid contains 13.6 percent antimony. It is supplied in ampules containing 6.4 percent solution (approximately 0.064 gm fuadin in 1 cc). Fuadin solution is given intramuscularly and should be injected slowly. The first three doses of 1.5 cc, 3.5 cc, and 5.0 cc are given on successive days. On the fifth and subsequent alternate days, 5 cc are given, provided no toxic effect other than nausea appears, until a total of 16 doses has been administered (75 cc of solution, containing 0.653 gm of antimony). The only commonly reported toxic symptoms are nausea and vomiting. Rarely, joint and muscle pains may appear. If toxic symptoms occur, the subsequent dose should be reduced or the administration temporarily or permanently discontinued, according to the circumstances. The results of treatment should be checked as described in paragraph 8. The course of treatment may be repeated after 2 weeks' rest. However, if a course of fuadin is ineffectual, it is recommended that the patient receive antimony and potassium tartrate.

d. *Antimony and potassium tartrate (tartar emetic)*. (1) This preparation contains about

36 percent antimony. It is recommended that it be administered in a concentration of 0.5 percent. Solutions should be freshly prepared, using if available Medical Department supplies of sterile 5 percent glucose in physiological saline solution, sodium chloride isotonic solution, or distilled water (preference in that order), rather than locally prepared diluent. If the diluent is prepared locally, it should be freshly distilled and sterilized. Every effort should be made to avoid the use of solutions containing pyrogens or the introduction of pyrogens during the preparation of solutions. Solutions of antimony and potassium tartrate should be perfectly clear and free of sediment. If a freshly opened clean supply of drug is available, with aseptic precautions it may be removed from the container, weighed, and transferred to sterile diluent, using sterile spatula and weighing vessels. With a fresh, clean supply of drug, sterile diluent, and careful technique, such a solution may be administered intravenously without sterilization. If doubt exists regarding the suitability of available supplies or technique, antimony and potassium tartrate solutions should be sterilized by gentle boiling for 5 minutes. They should not be autoclaved.

(2) Antimony and potassium tartrate is best tolerated 2 or 3 hours after a light meal. It should be administered intravenously and should be given slowly. Since the solution is very irritating to the tissues and may cause sloughing, the needle should be wiped off with a sterile sponge and there should be no extravasation. The patient should remain recumbent for at least an hour after treatment. The first dose of the 0.5 percent solution is 8 cc (0.04 gm tartrate). Provided no untoward reaction occurs, subsequent doses are given on alternate days and are increased on each occasion by 4 cc (0.02 gm tartrate), until 28 cc (0.14 gm tartrate) are being given. If no toxic reaction appears, a total of 15 doses is given (360 cc of solution, containing 0.648 gm antimony). The toxic effects of antimony and potassium tartrate include coughing immediately upon injection, which is not important; nausea; vomiting; stiffness of joints and muscles; sense of constriction of the chest; pain in the upper abdomen; bradycardia; dizziness; and collapse. Transient electrocardiographic changes with-

out corresponding clinical manifestations have been reported. If a toxic reaction other than coughing occurs during administration, the injection should be stopped at once. Following any toxic effect, the subsequent dose should be reduced or the administration of the drug temporarily or permanently discontinued, according to the circumstances. The course of treatment should not be repeated until after 2 weeks have elapsed without treatment.

8. FOLLOW-UP. In many cases, it is necessary to give more than one course of treatment. Hence, it is essential that the results of treatment for schistosomiasis be carefully checked. As chemotherapy becomes effective, eggs which are passed cease to contain viable miracidia. They appear dark, shriveled, and do not hatch when placed in water. When treatment is successful, eggs ultimately cease to appear. If viable eggs are present 4 weeks or more after a course of treatment is finished, it may be concluded that treatment has failed to kill all the worms present. Stool examinations should be done at least every week during treatment, at the end of a course of treatment, and at intervals thereafter for at least 3 months, but individuals should not be retained in the status of patients for this purpose alone. Individuals who have had schistosomiasis should be reexamined from time to time for a year following treatment. If eggs with living embryos are found, treatment should be reinstituted after a suitable interval has elapsed following the previous treatment.

9. PROGNOSIS. The proportion of patients with schistosomiasis japonica who acquire the late lesions of the disease is unknown. The amount and frequency of repetition of infection are important elements in determining the severity of the infection. The prompt administration of treatment has been found to relieve the early symptoms, including at least in some instances even severe manifestations of central nervous system involvement. It is believed that chemotherapy when instituted early is effective in preventing the development of severe sequelae. If cirrhosis of the liver is well established, chemotherapy does not influence the course.

10. PREVENTION. *a. General.* Prevention of schistosomiasis in military forces depends primarily upon avoidance of contact with fresh

water infested with cercariae. Control measures to interrupt the life cycle of the parasite by killing the snail intermediate hosts, or by preventing the pollution of water with feces containing eggs are of lesser immediate importance.

b. Avoidance of exposure. (1) Swimming, bathing, and laundering of clothes, and the washing of vehicles in fresh water ponds, streams, and canals should be prohibited in endemic areas. Surveys by trained personnel should be accomplished as early as possible to locate infested bodies of water, and appropriate warning signs should be posted. The apparent absence of snails does not guarantee freedom from cercariae. Educational lectures and propaganda posters should be freely utilized to indoctrinate troops in the necessity for avoiding contact with infested water. Orders regarding avoidance of exposure should be strictly enforced by the imposition of fines, or other appropriate punishment.

(2) When practicable, rubber hip boots, waders, rubber gloves, or other waterproofed clothing should be worn by personnel whose duties require that they work in unsafe water. Care should be taken not to splash water on unprotected parts of the body.

(3) Shower bath water for small units required to remain away from their base should be provided from Lyster bags in which the water is treated as described in *c* (2) below. The Lyster bag can be hung at a suitable height from a branch, or from a support stretched between two trees. Improvised showerheads should be prepared beforehand and connected to the faucets of the Lyster bag by a rubber hose. Use of such an expedient will minimize infractions of bathing discipline.

(4) In case of accidental or necessary entrance into water suspected of containing cercariae, immediate bathing with soap and brisk rubbing of the skin with a towel or article of clothing may reduce the chance of infection.

c. Treatment of water. Only water treated so that cercariae are killed should be used for bathing, laundry, vehicle washing, and drinking purposes. Maximum use should be made of subsurface water as a source of supply. Usual methods of filtration and chlorination cannot be relied upon to remove or destroy

cercariae. Recommendations for the treatment of water in areas where schistosomiasis is a hazard are summarized below.

(1) The standard portable (or mobile) sand filter purification units will not remove all cercariae; hence, sufficient chlorination to kill cercariae must also be employed. The newly standardized diatomaceous earth filter, however, is effective in filtering out cercariae.

(2) Application of chlorine sufficient to provide 1 part per million residual at the end of 30 minutes contact is sufficient to kill cercariae. It is important to note that this contact time exceeds that normally required for Lyster bag treatment. When employing Lyster bags, a satisfactory procedure which affords a margin of safety for the destruction of cercariae is to add sufficient calcium hypochlorite (grade A) to provide 1 part per million of residual chlorine after 10 minutes contact. An additional ampule of calcium hypochlorite should then be added and the water allowed to stand 30 minutes before use. When using canteens, treatment with two halazone tablets for clear water and four tablets for turbid or colored water is adequate to kill cercariae after 30 minutes contact.

(3) Water can be rendered free of infective cercariae by heating to 125° F. or by storage for [AG 300.5 (28 May 45)]

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72 hours. In either case, the water should be chlorinated before use.

d. *Destruction of snail hosts.* Copper sulfate or copper carbonate (3 pounds per 1,000 square feet of water surface to be treated) may be used to kill the snail intermediate hosts. A concentration of 10 parts per million of copper sulfate after 48 hours of contact destroys free-swimming cercariae as well as snails. Because of the amphibious habits of the snail hosts of *Schistosoma japonicum*, chemical treatment of the water should be supplemented by the application of slaked lime to the ground and vegetation on the banks and along the shoreline of the water. Measures for killing snails have their greatest usefulness where the infested bodies of water are small. Their value is limited in places where extensive areas must be treated.

e. *Disposal of feces.* Prevention of pollution of water by proper disposal of human feces from infected individuals is a control measure which reduces the number of infected snails. However, domestic animals such as dogs, cats, cattle, and water buffalo often harbor *Schistosoma japonicum* and may serve to spread the infection.